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Temperament: the bridge between biology and affective illness

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Kraepelin had envisaged a broad concept of manic-depressive illness that included recurrent depressions. The unipolar-bipolar dichotomy restricted the territory of manic-depression to strictly defined bipolar disorder with mania (bipolar I). Research over the past three decades has shown that bipolarity extends into the severe psychotic domain, as well as into the interface between bipolarity and unipolarity. At the severe end of the spectrum, familial-genetic and course parameters support the extension of bipolar disorder into "schizo-bipolar." At the "softer end," bipolar II is distinguished from bipolar I by excited periods which are non-psychotic and brief, and sometimes adaptive, hypomania as short as two days; in bipolar III, hypomania is associated with antidepressant treatment; in bipolar IV, the depression arises from a hyperthymic (trait subthreshold hypomanic) baseline. More recent data on the near normal distribution of hypomanic overactive behavior in bipolar II and unipolar patients is further evidence for the crumbling of boundaries between unipolar and bipolar disorders. The clinical and familial data in support for extending the bipolar spectrum has come from U.S. and European centers and community studies, and argues for oligogenic inheritance. However, the broadened clinical spectrum does not necessarily imply genetic homogeneity. The high population prevalence of bipolarity at the softer end of the spectrum (5–10%) argues for a role of bipolar traits such as cyclothymia and hyperthymia in human evolution (e.g. mate selection, territoriality, leadership, exploration, creativity). Finally, the broad spectrum has important therapeutic and public health significance in terms of early intervention and extending the benefit of mood stabilizers to conditions that might otherwise be diagnosed "unipolar" or "impulse control disorders."